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Supplemental Material

Effects of Crude Oil/Dispersant Mixture and Dispersant Components on PPARγ Activity *in Vitro* and *in Vivo*: Identification of Dioctyl Sodium Sulfosuccinate (DOSS; CAS #577-11-7) as a Probable Obesogen

Alexis M. Temkin, Robert R. Bowers, Margaret E. Magaletta, Steven Holshouser, Adriana Maggi, Paolo Ciana, Louis J. Guillette, John A. Bowden, John R. Kucklick, John E. Baatz, and Demetri D. Spyropoulos

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for *m/z* 421.0. Examining the fragmentation profile, noting the fragment ion *m/z* 81.0, indicates the presence of DOSS, as previously reported (Mathew et al. 2012; Ramirez et al. 2013).

Figure S3. Molecular modeling of PPARγ with COREXIT components Span 80, Tween 80 and dioctyl sodium sulfosuccinate (DOSS). A) Average E Score for COREXIT components analyzed for PPARγ ligand binding using MOE as in *Materials and Methods*. B) DOSS modeled in the PPARγ ligand-binding pocket.

Figure S4. PPARγ activity of COREXIT components other than DOSS. Dilutions of mixtures were prepared, HEK293T/17 cells were transfected, exposed in triplicate for 18 hours, and luciferase activities were measured as detailed in *Materials and Methods*. Dose-dependent ligand binding activities were not detected in any of the dilutions tested for A) Span 80 or C) ICP:Propylene Glycol (PG). Modest ligand binding activity was detected only at the highest dose of B) Tween 80 (50ppm). Robust dose-dependent ligand binding activities were detected in D) ICP:PG:DOSS (4 ppm and 8 ppm). Collectively, these results indicate that DOSS is the principle obesogen in COREXIT. Data are expressed as Mean ± SD; n = 3 per group (* $p < 0.05$ versus untreated controls).

Figure S5. DOSS has Human PPARγ, PPARα, PPARβ/δ, and RXRα transactivation activity. Dilutions of mixtures and positive controls were prepared, HEK293T/17 cells were transfected, exposed in triplicate for 18 h, and luciferase activities were measured. A) PPARγ, B) PPARα, C) PPARβ/δ and D) RXRα. Dose-dependent transactivation activities by DOSS were detected for PPARγ and PPARβ/δ. Data are expressed as Mean ± SD; n = 3 per group (* $p < 0.05$ versus not treatment control).

Figure S6. Dlk1 and Fabp4 mRNA expression in 3T3-L1 cells at day three of adipogenesis. 3T3-L1 cells were treated for 72 h as described in *Materials and Methods* and qPCR was performed as described in *Supplemental Materials and Methods*. Data are represented as a fold change in expression over the housekeeping gene HPRT for A) preadipocyte marker Pref-1/Dlk1 and B) adipocyte marker Fabp4 (* $p < 0.05$ versus MIM control, n = 6 per group).